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JACC FOCUS SEMINAR: MECHANICAL COMPLICATIONS OF ACUTE MYOCARDIAL INFARCTION, PART 2

JACC FOCUS SEMINAR

Ventricular Septal Rupture After Myocardial Infarction

JACC Focus Seminar 3/5

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ABSTRACT

Ventricular septal rupture remains a dreadful complication of acute myocardial infarction. Although less commonly observed than during the prethrombolytic era, the condition remains complex and is often associated with refractory cardiogenic shock and death. Corrective surgery, although superior to medical treatment, has been associated with high perioperative morbidity and mortality. Transcatheter closure techniques are less invasive to surgery and offer a valuable alternative, particularly in patients with cardiogenic shock. In these patients, percutaneous mechanical circulatory support represents a novel opportunity for immediate stabilization and preserved end-organ function. Multimodality imaging can identify favorable septal anatomy for the most appropriate type of repair. The heart team approach will define optimal timing for surgery vs percutaneous repair. Emerging concepts are proposed for a deferred treatment approach, including orthotropic heart transplantation in ideal candidates. Finally, for futile situations, palliative care experts and a medical ethics team will provide the best options for end-of-life clinical decision making. (J Am Coll Cardiol 2024;83:1886-1901) © 2024 the American College of Cardiology Foundation. Published by Elsevier. All rights reserved.

Ventricular septal rupture (VSR) remains a life-threatening mechanical complication of acute myocardial infarction (AMI). In the contemporary era, early revascularization strategies have effectively decreased the incidence of post-AMI VSR from ~2% to ~0.25%. Despite a remarkable decrease in disease prevalence, little have we affected the natural history and dismal prognosis associated with this condition.

The presenting features of VSR are closely related to the underlying pathophysiology.[1-5] In >50% of patients, septal rupture results in acute and progressive hemodynamic collapse and cardiogenic shock (CS). In these patients, conservative medical management is invariably fatal.[3,6,7] Corrective surgical repair, on the other hand, although potentially life-saving, is associated with high in-hospital mortality, particularly among unstable patients (>50%), with no

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HIGHLIGHTS

- The incidence of ventricular septal rupture after acute myocardial infarction has declined, yet there has been little improvement in clinical outcomes.
- Corrective surgery and percutaneous septal closure are options for patients with severe hemodynamic derangement.
- Collaboration among members of an interdisciplinary heart/shock team can help estimate the chances of viability vs futility, determine the role of mechanical circulatory support, and determine the timing of intervention.

improvement in mortality trends observed over the past few decades.[1,3,4,8] Transcatheter septal closure has emerged as a less invasive and life-saving alternative, with survival rates similar to those of corrective surgery.[1,3,4,8-13] As such, percutaneous closure should be considered as an up-front alternative in patients with favorable septal anatomy. Finally, recent iterations of mechanical circulatory support (MCS) devices, coupled with advanced heart failure programs, can offer a different pathway in the treatment of critically unstable or CS patients. In the opinion of the authors, this pathway represents a provocative opportunity in the management of these patients.[13-17] This *JACC* Focus Seminar revision provides a contemporary overview and novel perspective in the management scheme of patients with post-MI VSR.

INCIDENCE AND PREDICTORS OF VSR

VSR remains one of the most feared complications of a large transmural myocardial infarction (MI). In the prethrombolytic era, VSR was reported in more than 2% of all AMI cases.[2] The introduction of early thrombolysis and percutaneous coronary revascularization has resulted in a substantial decrease in VSR rates well below 1% (**Table 1**).[1,2,4-7,13,18] In the GUSTO-I (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) trial, VSR was reported in as few as 0.2% of patients and was attributed to improved reperfusion and myocardial salvage.[6] In the Apex-AMI (Assessment of Pexelizumab in Acute Myocardial Infarction) trial and GRACE (Global Registry of Acute Coronary Events) registry, the incidence of VSR was observed in 0.17% and 0.45%, respectively.[5,19] Similar rates were also reported in the MIDAS (Myocardial Infarction Data Acquisition System) database.[4] In patients with CS, the incidence of VSR is up to 5.8%, as reported in the SHOCK (SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK) trial registry.[20] Higher rates of VSR are associated with delayed revascularization, as observed during the COVID-19 pandemic.[21-37]

Several predictors and risk factors for VSR have been previously identified. These include advanced age, female sex, prior stroke, ST-segment elevation, anterior wall infarction, single-vessel coronary artery disease, high Killip class, and total coronary occlusion in the absence of collateral circulation.[1,4,6,18,19,38] In a study published by Vargas-Barrón et al,[39] persistent ST-segment elevation for >72 hours and right ventricular (RV) involvement were associated with increased risk of VSR. Negative predictors of VSR include pre-established cardiovascular risk factors such as hypertension, diabetes mellitus, prior MI, and peripheral artery disease. Ischemic preconditioning and collateral coronary circulation in these patients has been used to explain such findings.[1,4,6,19,38,40] Because the ventricular septum is supplied by the septal perforating branches, flow compromise of the left anterior descending or posterior descending coronary artery is more likely to result in VSR.[40]

ABBREVIATIONS AND ACRONYMS

AMI = acute myocardial infarction

CABG = coronary artery bypass graft

CS = cardiogenic shock

IABP = intra-aortic balloon pump

LV = left ventricle

MCS = mechanical circulatory support

MI = myocardial infarction

OHT = orthotopic heart transplantation

PCI = percutaneous coronary intervention

RV = right ventricle

VA-ECMO = venoarterial extracorporeal membrane oxygenation

VSR = ventricular septal rupture

PATHOPHYSIOLOGY AND CLINICAL PRESENTATION

The pathophysiology and clinical presentation of VSR may vary depending on the size, location, and selected treatment approach of the transmural infarct. Although most commonly seen during the first week following AMI, VSR presents with a bimodal distribution, peaking first during the first 24 hours and 5 days later, respectively.[3,6] In the present era of primary percutaneous coronary intervention (PCI), VSR occurs earlier and within the first day of an MI.[41] Similar changes have been observed with thrombolytic therapy, favoring hemorrhagic transformation and intramyocardial dissection rather than necrosis in the distribution of the infarct area.[6,42] The septal tear is exacerbated by the shear stress and preserved hypercontractile force of the adjacent myocardium infarct zone.[38,43]

The sudden onset of left-to-right ventricular shunting results in an abrupt rise in ventricular pressure and volume overload.[44] The aftermath is a rapid drop in forward cardiac output, systemic

TABLE 1 Morphologic, Pathophysiologic, and Interventional Characteristics According to the Anatomic Classification of Post-AMI VSR

	Apical	Anterior	Posterior	Basal
Morphology				
Simple/direct	+++	+++	+	-
Complex/serpiginous	-	+	+++	+++
Entity of the shunt				
Not hemodynamically significant	+++	+	-	++
Hemodynamically significant	+	+++	++	+
Massive	-	+	++	-
Ventricular involvement				
LV dysfunction	+	+++	+	+
RV dysfunction	-	-	+++	+
Biventricular dysfunction	-	+	++	++
Mitral valve involvement	-	-	++	+++
Hemodynamic impact				
Stability	+++	++	+	+
Instability	+	+++	++	++
Cardiogenic shock	-	++	+++	+
Cardiac arrest	-	+++	+	-
Pulmonary compromise				
No compromise	++	-	-	+
Pulmonary congestion	+	+++	++	+
Pulmonary edema	-	++	+++	++
Feasibility of repair				
Surgical	+++	+++	++	+
Percutaneous	-	+++	+	-

The mentioned pathophysiologic and interventional considerations are directly influenced by the size of the rupture. + = mildly present; ++ = moderately present; +++ = highly present.
AMI = acute myocardial infarction; LV = left ventricular; RV = right ventricular; VSR = ventricular septal defect.

hypotension, oliguria, and CS, observed in ~50% of patients.[8,44] Notwithstanding, endogenous sympathetic systemic compensatory mechanisms lead to vasoconstriction, increasing afterload, left ventricular (LV) wall tension, and worsening left-to-right shunt. As the insult progresses, acute pulmonary congestion and RV failure ensues—an important negative predictor of outcome.[13,14,44]

Depending on the magnitude of the septal defect, the clinical presentation of a post-AMI VSR may vary from mild dyspnea to fulminant circulatory collapse and cardiac arrest.[13,20,44] VSR should be suspected in any patient with acute-onset shortness of breath, chest pain, pulmonary edema, hypotension, and/or shock days soon after an AMI. The presence of a loud, harsh holosystolic murmur with a palpable thrill should guide clinicians to the suspicion of VSR.[13,44] A high index of suspicion is required to confirm the diagnosis.[7,13,38] Other forms of post-MI mechanical complications should be excluded. In the case of acute pulmonary edema, it is perhaps more commonly observed in patients with papillary muscle rupture, whereas free wall rupture more often presents with acute cardiac tamponade in the absence of a murmur.[45] Biventricular failure, on the other hand, may be seen with large septal defects.[45]

CLASSIFICATION

VSR may be classified according to location as an apical, anterior, or posterior VSR (**Table 1**).[38,44] Apical and anterior defects are most common and typically result from AMI involving the left anterior descending artery. Posterior VSR is more commonly seen in patients with right coronary artery involvement and has been associated with a higher incidence of biventricular dysfunction.[3,6,13,38,43,46,47]

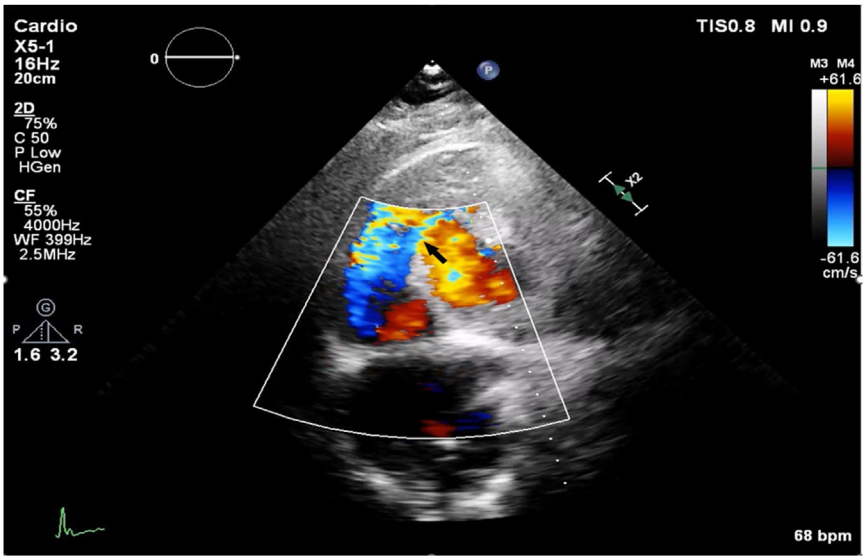
Additionally, VSR may be categorized according to its morphology as simple or complex.[47] In simple defects, the rupture is direct, through and through across the RV and LV. In complex defects, multiple rupture sites and a serpiginous tract may be seen.[44] Although not clearly understood, complex defects have been more commonly described in posterior septal defects, and in cases of intramyocardial hemorrhage or intramural dissecting hematoma.[6,13,44,47]

DIAGNOSIS AND MULTIMODALITY IMAGING

Transthoracic echocardiography represents the initial best imaging modality for patients with suspected VSR and is recommended as a Class I, Level of Evidence: C for the diagnosis of AMI-related mechanical complications.[48] Assessment of the left-to-right shunt across the interventricular septum may be obtained using color-flow Doppler examination (**Figure 1**), and 2-dimensional imaging allows for characterization of defect size; location; and morphology, including assessment of biventricular function.[38,45,49,50] In patients with VSR, the noninfarcted myocardium may appear hyperkinetic, whereas the RV and pulmonary artery may appear dilated from the acute volume overload.[43] Other forms of mechanical complications may be excluded, including free wall rupture or papillary muscle rupture, making early echocardiographic assessment important for such distinction. Transesophageal echocardiography is often used to confirm the diagnosis and further characterize the septal defect, particularly in patients with poor acoustic windows and those in whom a VSR has not been clearly excluded.[38] Its use is equally valuable during intraoperative imaging and procedural guidance.

Recent advancements in cardiac magnetic resonance and high-resolution computed tomography have substantially elevated the ability to assess myocardial function, intracavitary volumes, congenital or acquired intracardiac defects, and myocardial tissue characterization. Novel postprocessing 3-dimensional

FIGURE 1 Echocardiographic Examination of Ventricular Septal Rupture



Echocardiographic examination with color flow Doppler showing left-to-right turbulent flow across the interventricular septum following acute myocardial infarction (black arrow).

reconstruction techniques have also been uniformly valuable in the management of complex valvular and structural heart disease.[51,52] In post-MI VSR, the use of multimodality imaging has been proven fundamental to guide clinical decision making and preprocedural planning.[51,52] The greatest limitation revolves around access and ease of use, particularly in critically ill patients.[50,53,54]

Finally, a low threshold should exist to perform a right heart catheterization to confirm the diagnosis and for hemodynamic profiling. Assessment of intracardiac filling pressures, interventricular shunting, estimation of RV function, and cardiac output is paramount in the critical care of these patients.[13,44,55]

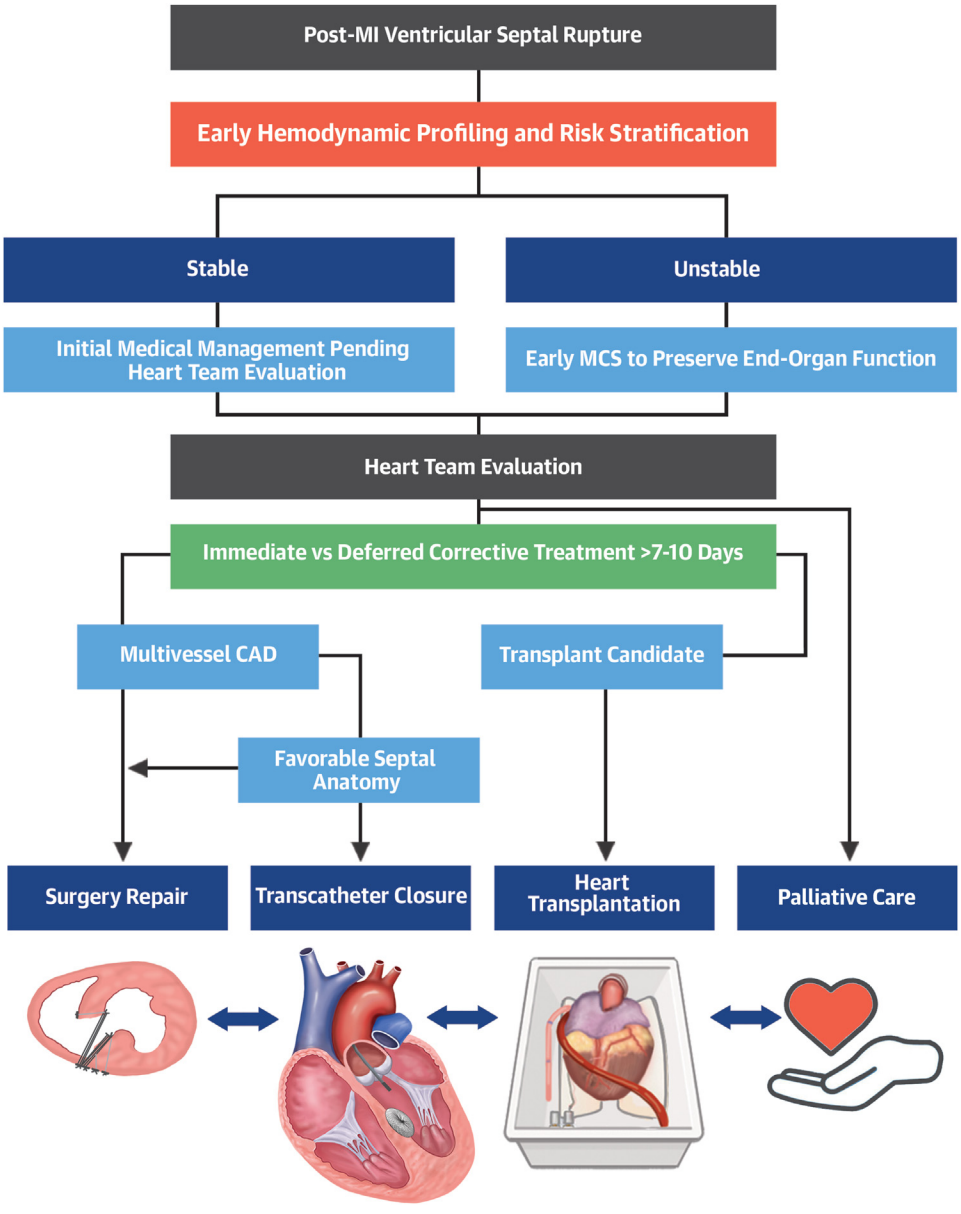
CLINICAL PRACTICE GUIDELINES

Guideline recommendations for patients with VSR stem from the American College of Cardiology and American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology guidelines.[48,55-57] In the previous U.S. guidelines dating back to 2013, early corrective surgery was considered paramount, even among stable patients at risk for VSR expansion and hemodynamic collapse.[56] In the updated guidelines from 2021, the investigators highlight a lack of consensus on optimal timing for surgery, recognizing that a higher mortality rate may be expected with early repair and that perhaps a deferred approach, particularly among those responding well to early aggressive management, may be more beneficial.[57]

The recent update also incorporates the emerging value of temporary MCS during preprocedural hemodynamic stabilization while acknowledging the absence of robust scientific evidence to determine its true clinical benefit.[48] Another important update includes the incorporation of transcatheter VSR closure as an alternative to surgery, particularly among patients with favorable septal anatomy, at prohibitive surgical risk, and temporizing measure to corrective surgery (Class I, Level of Evidence: C).[48] Finally, the importance of a multidisciplinary team-based approach with shared decision making is highlighted, as also acknowledged by the 2021 American Heart Association expert consensus statement on post-AMI mechanical complications[48,57] (**Central Illustration**).

LIMITATIONS OF THE GUIDELINES. It is pivotal to understand that the existing guidelines on VSR management are sparse and generally limited by the absence of high-quality evidence. The recommendations largely stem from unfounded scientific evidence, including the opinion of expert leaders, and are challenged by the acuity, complexity, and low VSR prevalence rates and associated high mortality. The limitations are best represented by the large

CENTRAL ILLUSTRATION **Contemporary Treatment Scheme of Ventricular Septal Rupture Following Myocardial Infarction**



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A proposed contemporary algorithm for the treatment of patients with postmyocardial infarction ventricular septal rupture. The importance of an early risk-stratification strategy to identify those with stable vs unstable hemodynamics is shown. The early use of novel mechanical circulatory support devices to stabilize and preserve end-organ function is represented. The importance of a heart team approach and the potential role of OHT in selected candidate patients with cardiogenic shock is underscored. The value of advanced multimodality imaging is further incorporated to further risk-stratify patients who could potentially benefit from transcatheter closure over open surgical repair, irrespective of the hemodynamic status. Finally, the interplay of multivessel coronary disease and the option of palliative care are expressed across the continuum of the algorithm. CAD = coronary artery disease; MCS = mechanical circulatory support; MI = myocardial infarction; OHT = orthotopic heart transplantation.

interinstitutional variations observed in the care of these patients, conditional to the local expertise and available resources.

MANAGEMENT

MEDICAL MANAGEMENT AND PREOPERATIVE EVALUATION. Despite ongoing efforts to provide updated guideline recommendations, the natural history and prognosis of VSR have remained steady over the years. The malignant course of VSR typically starts with a precipitous insult that often results in abrupt hemodynamic collapse. In CAUTION (Mechanical Complications of Acute Myocardial Infarction), ~50% of patients with VSR developed CS. Early recognition and hemodynamic stabilization to reduce the risk of circulatory collapse and multiorgan failure are the cornerstones of management.[8] Although VSR repair is ultimately required, initial medical therapy should be targeted at effectively reducing ventricular afterload with the aim of decreasing the magnitude of left-to-right shunt, preserving antegrade flow, maintaining hemodynamic stability, and supporting the ischemic myocardium.[48,58]

Early risk profiling with a Swan-Ganz pulmonary artery catheter should be encouraged for hemodynamic characterization, treatment selection, and subsequent medication titration (Class IIb, Level of Evidence: B).[55] The pulmonary artery catheter is particularly valuable in guiding escalation or de-escalation of medical therapy and MCS. Furthermore, it offers the ability to tailor therapy and trends in shunt flows via oxygen saturation analyses. A multitude of hemodynamic parameters, including right and left filling pressures, pulmonary and systemic shunt flow ratios, cardiac output and index, pulmonary artery pressure, pulmonary vascular resistance, and other correlates of RV function such as the pulmonary pulsatility index, may be derived from its use to guide management.

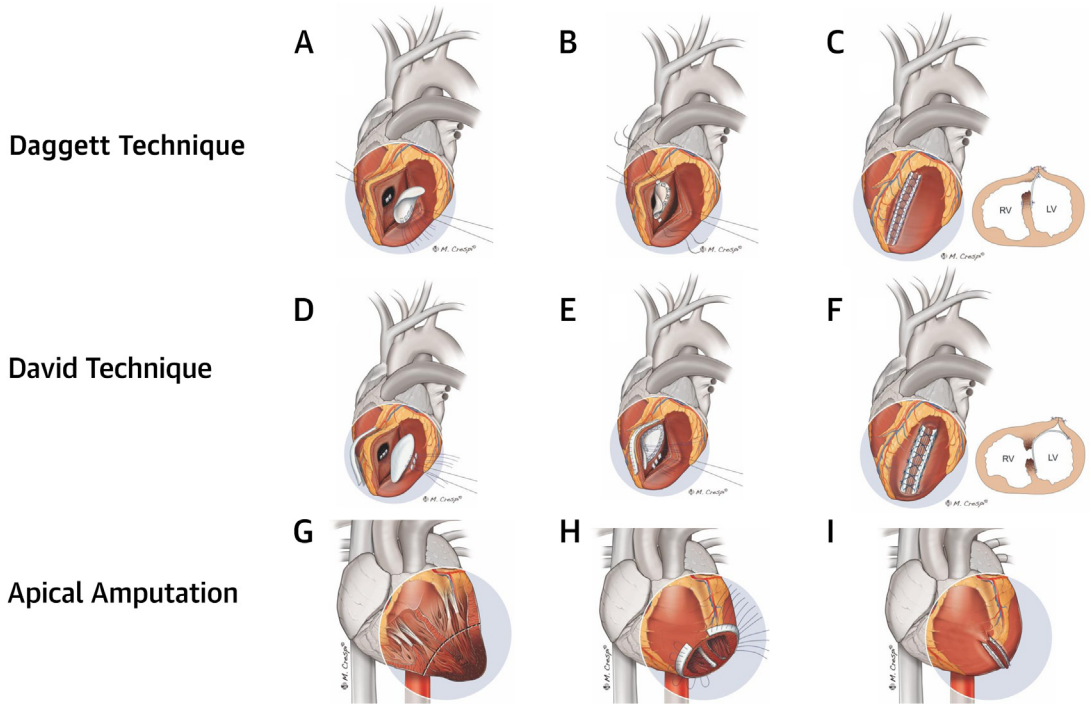
In unstable patients, medical treatment alone has never been shown to improve outcomes in VSR patients and may negatively affect patient survival by potentially delaying corrective surgery in cases of refractory CS.[6,13] The use of intravenous vasodilators to reduce systemic afterload has never been clearly validated and may precipitate hemodynamic collapse. In stable patients, supportive medical therapy, however, may allow for a deferred corrective treatment strategy, which has been associated with favorable outcomes.

MECHANICAL CIRCULATORY SUPPORT. In VSR-CS patients, timely implementation of MCS may reduce the detrimental escalation of inotropic and vasopressor therapy. In the opinion of the authors, up-front use of MCS coupled with rigorous shock team protocols provides an opportunity to potentially amend the prognosis and natural history of VSR shock.[14,43,59-63] The benefits of an early MCS strategy as a bridge to definitive therapy are limited but have been previously reported with the use of Impella (Abiomed), TandemHeart (LivaNova), and venoarterial extracorporeal membrane oxygenation (VA-ECMO).[11,14,63,64] Novel MCS devices offer the ability to reduce VSR-mediated shunt flow by decreasing LV afterload, preserving forward cardiac output and systemic organ perfusion (**Figure 2**).[14,58,61,65] In the latest European Heart Failure guidelines as well as the 2023 updated acute coronary syndrome guidelines, the up-front use of MCS before corrective surgery is encouraged as the initial intervention for patients with acute decompensation or CS following post-AMI complication (no level of evidence recommended).[48,66]

Understanding the individual MCS properties is fundamental when deciding on the most appropriate strategy. Avoiding maladaptive MCS physiology, particularly in patients with intracavitary shunts, is critical.[14,67] Device selection should therefore be tailored to the mechanisms of ventricular failure, operator experience, and institutional proficiencies.

In the case of the counterpulsation intra-aortic balloon pump (IABP), it has been shown to reduce LV afterload, increase coronary perfusion, and decrease myocardial wall stress. Its low cost and ease of use make it convenient and explain its global adoption.[14,58] In the SHOCK trial registry and the Society of Thoracic Surgeons national database, IABP was used in 75% and 65% of VSR patients, respectively.[9,14,20,55,58,60] In the recent European guidelines, IABP should be considered to decrease ventricular afterload in patients with hemodynamic instability or CS caused by AMI-related mechanical complications (Class IIa, Level of Evidence: C).[48] When compared to other forms of MCS, IABP may not effectively unload the LV or enhance cardiac power output.[68] In the IABP-SHOCK trial of patients with AMI-CS, the routine use of IABP failed to improve survival when compared to medical therapy.[69] Other devices, including the percutaneous microaxial transaortic devices (eg, Impella) have been proven more beneficial in these circumstances.[14] Depending on the size of the cannula and mode of insertion (surgical or percutaneous), the Impella microaxial catheter pump can deliver up to 3.5 to 5.5 L of flow. In patients with VSR, its use has been shown to be safe and effective in reducing ventricular shunting and pulmonary venous congestion as well as

FIGURE 2 Most Common Surgical Techniques for Postinfarction Ventricular Septal Defect



(A to C) The infarct excision technique described by Daggett et al.[79] (D to F) The infarct exclusion technique described by David et al.[80] (G to I) The apical amputation technique described by Daggett. See text for details. LV = left ventricle; RV = right ventricle.

improving gas exchange while reversing the maladaptive hemodynamic detriments.[13,14,68,70,71]

In cases of profound VSR-CS with severely compromised multiorgan failure, total cardiopulmonary bypass support via percutaneous VA-ECMO may be necessary to enable end-organ recovery before definitive surgery. Contingent to the hemodynamic status, ECMO cannulation may be performed readily at the bedside under fluoroscopy or transesophageal echocardiography guidance. As ECMO flows progressively increase, so does the LV afterload and the degree of left-to-right shunting.[14,61,65] In these cases, the adjuvant use of percutaneous or surgical LV vents (ie, Impella) may be beneficial to reduce pulmonary shunt fraction and minimize the risk of acute lung injury.[14,70,71] Furthermore, LV venting increases LV stroke volume and reduces the inherent risk of LV or aortic thrombosis seen with ECMO. The benefits of MCS should, however, be weighed against its potential harm.[72] In the case of ventricular microaxial pumps, aspiration and embolization of necrotic tissue debris, septal wall trauma, and right-to-left shunt reversal resulting in systemic hypoxemia should be considered.[14,62,72,73] Finally, vascular injuries represent another major source of MCS-related complication, as emergent cannulations are often necessary.[74]

The lack of scientific evidence to foster and support a universal algorithm that incorporates an early aggressive MCS strategy as a primary treatment strategy in patients with post-MI VSR limits any meaningful conclusion or recommendation. In a recent multicenter study published by Thiele et al,[74] the up-front use of VA-ECMO in patients with AMI and CS provided no survival benefit at 30 days. Notably, the investigators specifically excluded patients with mechanical complications of AMI. This population may benefit from the early use of temporary MCS, preserving end-organ function and allowing for a more timely corrective treatment, according to the heart team approach.[14,65] In a recent observational, retrospective, multicenter study by Sanchez Vega[11] conducted in Spain, a progressive trend toward increased use of VA-ECMO resulted in greater access to corrective treatments, with higher survival rates. The ease of use of MCS facilitates its early implementation and enables smaller hospital systems without local expertise the opportunity to stabilize

and transfer patients to tertiary centers with broader experience in surgical or percutaneous management, including durable MCS and orthotopic heart transplantation (OHT).[11,15,16,75-77]

SURGICAL TREATMENT AND TECHNIQUES. VSR was once considered an inoperable condition.[13] In 1957, Dr Denton Cooley successfully performed the first surgical VSR repair.[78] Since then, various techniques have been proposed, using different approaches depending on the localization and type of rupture. **Figure 2** summarizes 3 of the most commonly used techniques. Of these, the "infarct excision" technique or primary repair, described by Daggett et al (**Figures 2A to 2C**), and the "infarct exclusion" technique, described by David et al (**Figures 2D to 2F**), with their subsequent modifications are used most frequently.[79-81]

In the case of anterior septal defects, the infarcted surface of the LV is incised parallel to the left anterior descending artery. For posterior septal defects, a ventriculotomy is performed in the affected posterior LV wall, parallel to the posterior descending coronary artery. In a true apical defect, the apical amputation technique described by Daggett et al[79] may be used. This involves a ventricular apical amputation that completely excludes the septal communication. This technique has also been reported to provide satisfactory results (**Figures 2G to 2I**). Primary closure of the left ventriculotomy is often possible with mattress sutures buttressed with pericardium or felt, reinforced with continuous sutures and BioGlue.[44,79]

Various other techniques have been reported to address several technical downsides of the previous approaches, including those related to patch dehiscence resulting in residual or recurrent VSR.[82] The use of more than 1 pericardial patch is now favored to ensure a successful VSR repair and to sustain ventricular pressure.[83,84] In a recent meta-analysis comparing alternate strategies, a trend toward lower operative mortality was observed with the infarct exclusion technique, although this did not reach statistical significance.[3,85]

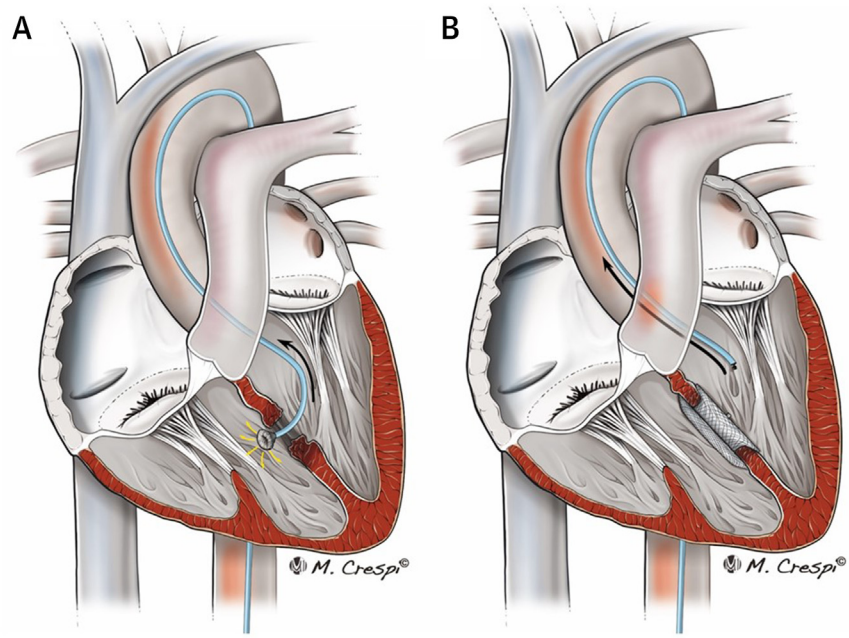
Equally important to the surgical techniques is the postoperative management of VSR repair.[82] Most causes of death among successfully repaired VSR involve patients with severely impaired univentricular or biventricular failure and their direct sequala (ie, CS and multiorgan failure).[13] In cases of refractory shock, postoperative management with MCS support is critical to improve patient outcomes.[14,61,65] In this critical phase, MCS may allow for adequate LV afterload and wall tension reduction as well as protection against suture dehiscence and VSR recurrence.[14] The importance of aggressive perioperative management after VSR repair has been supported by recent reports showing an increasing adoption of MCS in this setting.[14,59,65,86,87] In the CIVIAM registry, a significant increase in MCS adoption was observed within the 5-year period of 2013 to 2018 when compared to the previous period.[11]

CORONARY REVASCULARIZATION. Coronary revascularization at the time of VSR repair has long represented a matter of debate.[20,88,89] In the case of PCI, treatment of the culprit artery has been generally discouraged because it is presumed that the infarct territory involving the VSR has been completed.[88] The addition of antiplatelet therapy required post-PCI also represents a dilemma by increasing the risk of perioperative bleeding at the time of VSR repair. Similarly, findings have been observed with patients undergoing concomitant coronary artery bypass grafting (CABG).[88] In a meta-analysis of 4,312 patients with post-AMI mechanical complications (mostly VSR related), 49% underwent concomitant CABG with little to no benefit observed during early and late postoperative mortality.[88] Moreover, in a study published by Horan et al,[90] a higher mortality trend was observed with concomitant CABG, albeit not significant during long-term follow-up.

Concomitant revascularization of the nonculprit territories in patients with multivessel disease, on the other hand, may actually be beneficial because it is believed to protect the residual myocardium at risk, resulting in a possible survival benefit.[88,91-95] Understanding whether single vs multivessel coronary disease is present at the time of VSR repair may therefore be important.[8,41,88,95-101] In the 2021 American College of Cardiology/American Heart Association/Society for Cardiovascular Angiography & Interventions consensus guidelines for coronary revascularization, concomitant CABG at the time of VSR repair was deemed appropriate as a Class I indication, Level of Evidence: B.[57] Conversely, the role for single-culprit artery revascularization was less favored, having no apparent effect on early or late mortality.

TRANSCATHETER CLOSURE. The combination of acute left-to-right shunting in a weakened infarcted myocardium, added to the intrinsic offense of open heart surgery, is known as the "triple insult."[9-11,38] Transcatheter closure techniques have recently emerged in an effort to overcome the high

FIGURE 3 Transcatheter Closure of Ventricular Septal Rupture: Retrograde Approach



(A) The catheter across the aortic valve and through the ventricular septal defect. (B) The Amplatzer (Abbott) delivery catheter is exchanged over the wire into the RV chamber, through which an Amplatzer Occluder is deployed and released to close the septal defect. Reproduced with permission from M. Crespi.

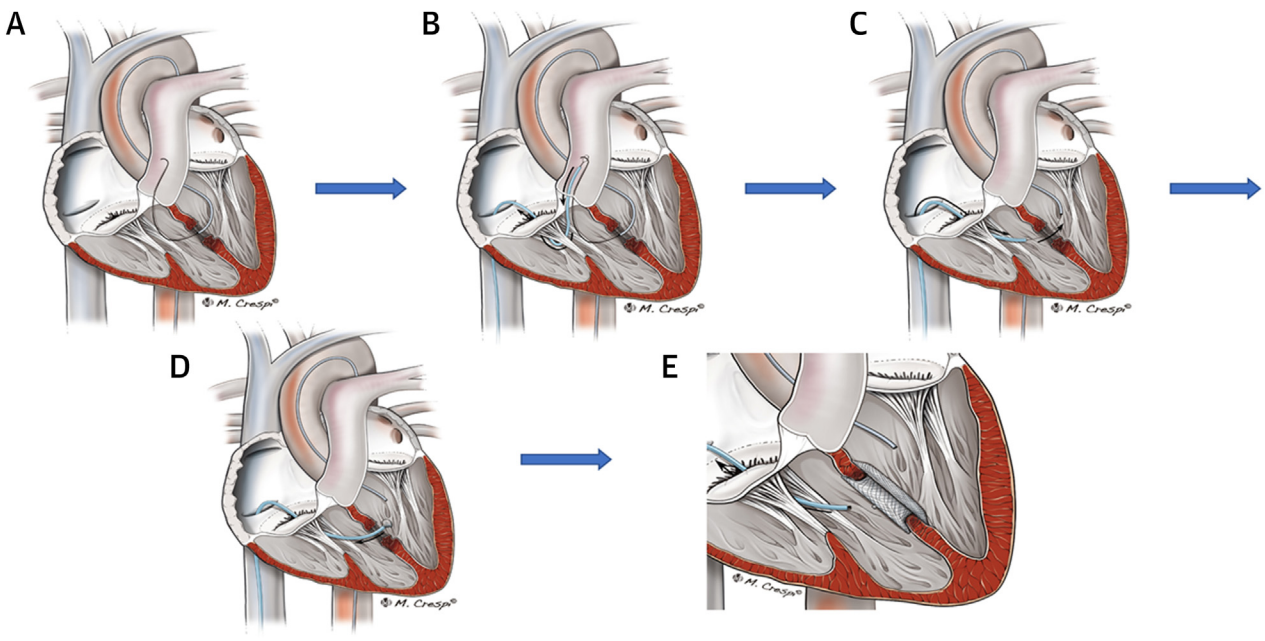
perioperative risk and mortality associated with surgical VSR repair.[10,13,14,44] A diversity of off-label transcatheter closure devices have been previously used for this purpose. The VSR-specific Amplatzer Post-Infarction Muscular Ventricular Septal Defect Occluder (Abbott Vascular, Inc) represents one specifically designed for this condition.[102-104]

Although initially described as a palliative measure for patients deemed not suitable for surgery, expertise in transcatheter closure has resulted in increasing adoption in selected patients with favorable anatomy.[103] The advent of multimodality imaging, including 3-dimensional printing technology, high-resolution cardiac computed tomography, and intracardiac echocardiography, have heightened the safety and efficacy of transcatheter closure, supporting a tailored approach to device selection.

Transcatheter closure is most commonly performed under general anesthesia using intraoperative transesophageal echocardiographic and fluoroscopic guidance.[44] Device selection is highly variable and often conditional to operator preference; availability; and VSR size, location, and morphology. Similarly, different techniques have been reported, including retrograde, antegrade, transseptal, and hybrid periventricular approaches. In the case of a retrograde approach (Figure 3), transcatheter closure is performed following arterial-femoral cannulation with retrograde access across the aortic valve (Figure 3). The steps involved with this technique are summarized in Figure 4. The VSR may be engaged with a selective use of 5- to 6-F Judkins right coronary catheter and a hydrophilic wire to transverse the defect into the RV and main pulmonary artery (Figure 4A). The wire is snared and exteriorized through a contralateral femoral or jugular venous approach creating an arterial-venous loop to enhance guide-catheter stability and effective closure of the septal defect.[10,13]

In the antegrade approach (Figure 5), the septal defect is engaged in a similar fashion following right femoral venous puncture, across the tricuspid valve and into the LV from the RV. This technique may be particularly valuable for patients with mechanical aortic valve prosthesis. A modified retrograde variant that incorporates a transseptal puncture may be required in the rare instance of an aortic and tricuspid mechanical prosthesis to create a venous-venous wire loop.[13,105] A hybrid periventricular approach (Figure 6) may also be considered in selected cases.[106] Finally, the bridge across septum technique using sutures in a nonendoluminal trajectory (BASSINET)

FIGURE 4 Transcatheter Closure of Ventricular Septal Rupture: Retrograde Approach



(A) The catheter and wire are advanced in a retrograde fashion across the aortic valve and through the ventricular septal rupture. The wire is then advanced into the main pulmonary artery. (B) The catheter and snare device are advanced in an antegrade fashion into the main pulmonary artery to snare the wire. (C) The wire is snared and exteriorized, creating an arterial-venous loop. (D) The Amplatzer delivery catheter (Abbott) is advanced antegrade over the wire and across the tricuspid valve through the defect. (E) The Amplatzer Occluder is deployed and released to close the septal defect. Reproduced with permission from M. Crespi.

procedure previously reported by Kamioka et al combines a subxiphoid surgical approach with transcatheter device closure, using 2 Amplatzer Cribriform Septal Occluder (Abbott) devices, reducing surgical risks in cases of difficult anatomy.[104,107]

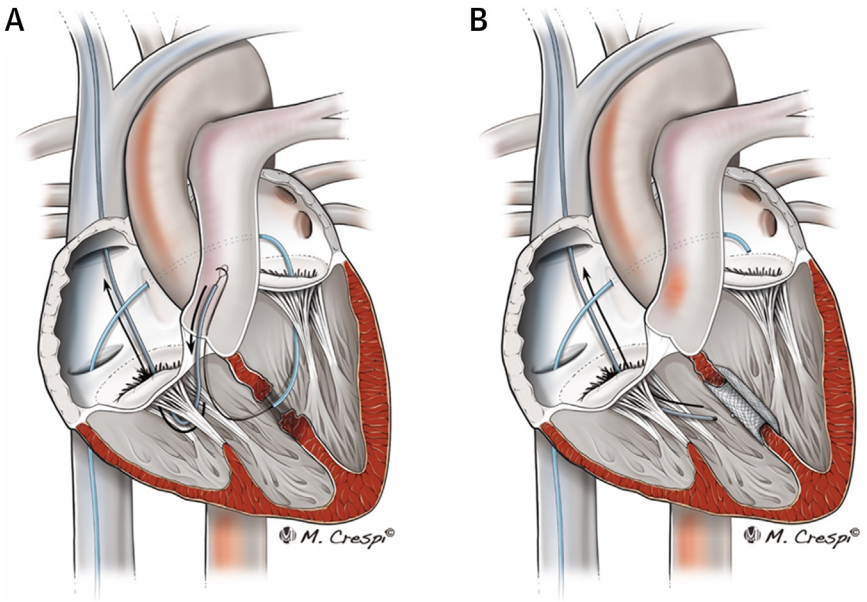
Complications related to transcatheter closure include cardiac arrhythmias, residual VSR, and ventricular perforation. The surrounding fragile infarct tissue may also predispose patients to device embolization and migration.[13] The latter may be obviated with device oversizing, but this too increases the risk of impairing adjacent structures such as the tricuspid valve.[44] Careful case selection and procedural planning are paramount; hence, the procedure is often technically challenging, requiring a high level of catheter dexterity. A potential advantage of transcatheter closure is that it does not preclude the option of subsequent surgical repair.

OUTCOMES AND PREDICTORS OF SURGICAL AND TRANSCATHETER CLOSURE

SURGICAL REPAIR. Despite advancements made in surgical closure techniques, the expected in-hospital mortality rate for patients with VSR remains steady and exceeds 30% to 60%.[11,108] In a recent meta-analysis of post-MI VSR involving 6,361 adult patients from 41 studies, the overall operative mortality was 38.2%. In the same study, the investigators found no difference in operative outcomes among those treated between 1971 to 2000 and 2001 to 2018.[3] In unstable patients with VSR-CS, the mortality associated with surgical repair may exceed ~80%.[9] For many, it is considered one of the most lethal cardiac surgical procedures.[3,8,9]

Recommendations related to the timing of surgery have remained subject to great controversy. In a study published by Arnaoutakis et al[9] involving 2,876 patients with VSR, the investigators report an overall 30-day mortality of 42.9%, with a significant decrease in mortality to 18.4% after 7 days vs 54.1% within 7 days of an AMI. Delay in surgery has been proposed to facilitate successful repair by allowing friable tissue to organize, strengthen, and become well differentiated from surrounding healthy tissue. The findings must be interpreted with caution, because patients undergoing delayed surgery may be naturally stable enough to bear the condition, resulting in

FIGURE 5 Transcatheter Closure of Ventricular Septal Rupture: Antegrade Transseptal Approach



(A) The catheter and wire are advanced in an anterograde fashion from the femoral vein and across the interatrial septum, through the mitral valve into the ventricular septal rupture. The wire is then advanced into the main pulmonary artery. The next steps are the same as with the retrograde technique displayed in Figure 4. Briefly, the wire is then snared in the main pulmonary artery and exteriorized to create a venous-venous loop. This is done using a second femoral venous access point or the right jugular vein, as shown in this example. (B) The Amplatzer delivery catheter is advanced over the wire and across the tricuspid valve from the right jugular vein. The Amplatzer Occluder is deployed and released to close the septal defect. Reproduced with permission from M. Crespi.

possible selection or survival bias.[8,9,14,61,87] Other explanations include optimal patient selection and lower risk of perioperative bleeding associated with antiplatelet therapy washout.[9,13]

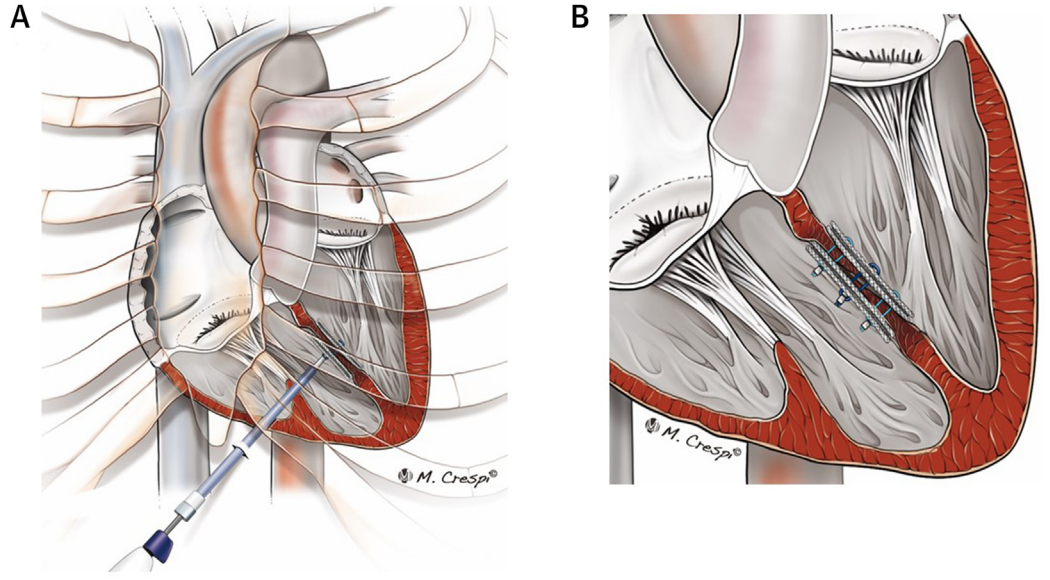
Several predictors of VSR surgical outcomes have been previously identified. These include, unfavorably, the presence of biventricular failure, high Killip class, inferior AMI, posterior VSR, inotropic support, MCS, emergent surgery, and total occlusion of the infarct-related artery.[3,6,9,44,109] In CAUTION, older age, cardiac arrest on admission, PCI, and postoperative IABP or ECMO support were also associated with worse outcome.[8] Conversely, longer times from AMI to VSR occurrence and from VSR diagnosis to surgical repair have been associated with lower in-hospital mortality.[4,9]

TRANSCATHETER CLOSURE. Outcomes associated with transcatheter VSR closure compare similarly to those of surgical repair.[110] In a systematic review of transcatheter VSR closure, successful device deployment occurred in 89%, with overall in-hospital mortality of 32%. The review included 48% of patients who at the time of their procedure were in CS.[111] In another meta-analysis, Yang et al[12] observed a 31.5% mortality 30 days after transcatheter VSR closure. In a study published by Giblett et al,[10] up to 16.1% of patients undergoing transcatheter closure required subsequent surgical repair, and another 7.8% required repeat transcatheter intervention.

In the same study, the in-hospital mortality was significantly lower for the surgical group (44.2% vs 55.0%), though no differences in long-term mortality were observed (53.7% vs 61.1%). Independent predictors of long-term mortality included CS, a transcatheter approach, and number of diseased coronary arteries.[10] Additional negative predictors of transcatheter outcomes include delayed diagnosis, liver failure, large left-to-right shunt at time of closure, and residual postprocedural shunt.[13,112]

Despite its poor prognosis, in the contemporary era of VSR management, surgical repair should be aggressively considered by the heart team approach. According to the European CIVIAM registry, published in 2021, up to 70.3% patients with VSR underwent surgical attempt, whereas transcatheter closure was performed in only 6.8% of cases.[11] The

FIGURE 6 Hybrid Transcatheter-Periventricular Approach



(A) Following percutaneous or surgical exposure (via small surgical window), a catheter is advanced through the right ventricular free wall and across the ventricular septal defect. (B) The Amplatzer Septal Occluder is deployed and released to close the septal defect. Reproduced with permission from M. Crespi.

decision to pursue surgery vs transcatheter closure varies significantly depending on local expertise, hemodynamic status, and anatomic characteristics.[13,44,103] Not infrequently, complex serpiginous defects, apical defects, and those adjacent to the valvular apparatus make transcatheter closure a less favorable option. Finally, studies directly comparing one strategy over another are unlikely to occur, given the complex scenario, acuity of care, and low disease prevalence.[10,12,102-104,110]

ORTHOTROPIC HEART TRANSPLANTATION. Considering the controversial outcomes of surgical and percutaneous closure, a thoughtful consideration for alternate options is necessary.[3,8] Improvement in access and surgical outcomes with OHT should be viewed as an opportunity to review and deviate from the traditional strategy of surgical or percutaneous repair, which carries close to a 90% mortality rate in patients with CS. Isolated case reports of OHT in patients with VSR have described survival rates remarkably superior to those of conventional treatment.[3,13,15] In a retrospective multicenter study of 120 patients with VSR, 5 of 6 patients (~84%) who underwent OHT survived their index hospitalization.[11] In another systematic review of 17 patients with VSR undergoing OHT between 1995 and 2015, the average in-hospital mortality was 29%, including patients deceased while waiting for OHT.[15] In younger, otherwise healthier individuals with post-MI VSR, OHT may offer a high likelihood of survival (**Central Illustration**). Local access to advanced heart programs represents the biggest challenge. Not only are there a limited number of transplant centers, but these are often constrained by financial, regional, and national variations related to transplant policies, including long waiting times and organ availability. Furthermore, the imbalance between donors and recipients mandates careful evaluation to avoid improper listing of VSR patients beyond those in whom OHT represents the only available option.

PALLIATIVE CARE. Studies evaluating the impact of a shared decision-making strategy, orchestrated by a critical care physician, have been shown to enhance collaboration, streamline transition of care, and improve patient survival.[13,113-115] Collaborative care teams should include nurses, respiratory therapists, pharmacists, interventional cardiologists, cardiovascular surgeons, heart failure clinicians, social workers, dieticians, and others. Equally important is the participation of palliative care experts and a medical ethics team to help guide physicians and identify patient-centered goals of care and assistance in end-of-life discussions. In VSR-CS, clinicians are tasked with the fine balance of aggressive

intervention vs palliative care, which in many instances represents the best alternative for a given patient and family. Patients and family members should therefore be integrally involved in the complex care and clinical decision-making process[113,115] (Central Illustration).

LONG-TERM OUTCOMES

With regard to the long-term operative outcomes of VSR, a study from New Zealand reported actuarial survival for surgically treated VSR patients of 66% at 1 year, 62% at 3 years, and 58% at 9 years. The study findings suggest that after the initial critical period, late survival is satisfactory.[116] Similar findings have been asserted in the CAUTION study. In that study, the 5- and 10-year survival rate was 80.2% and 74.4%, respectively, after hospital discharge, showing VSR to have the highest expected long-term survival among mechanical complication types.[117] The result suggests that once through the critical perioperative phase, life expectancy may not be inferior to that of other AMI patients with LV dysfunction.[118] The Swedish Registry reported a cumulative survival of 38% at 5 years but up to 67% in those discharged alive.[91] Subsequent studies reported 5-year and 10-year survival rates approaching 70% to 80% and 40% to 50%, respectively.[119,120] Other than emergency status, older age and concomitant CABG were independently associated with worse outcome at 1 year.[120] Poor long-term survival was associated with RV dysfunction, residual LV dysfunction after surgery, and elevated NYHA functional class.[119]

FUTURE PERSPECTIVES

Despite advancements in the management of AMI, post-AMI VSR remains a rare but extremely severe condition whose outcomes have changed little in the last decades. Given the complexity of the condition, risk stratification is urgently needed. A classification based on cardiac function, hemodynamic stability, anatomic variations, and multiorgan involvement may offer personalized treatment algorithms aimed to improve survival.

If the heart team considers that delayed repair may improve in-hospital outcomes, all necessary measures should be implemented to successfully postpone repair. All modalities of MCS should be considered, providing a tailored combination of devices that fits the individual patient. Moreover, in patients with favorable septal anatomy, transcatheter VSR closure should be pursued in lieu of surgical repair, according to prespecified characteristics. Further evidence is needed to provide information regarding the most effective algorithms to treat post-AMI VSR patients, ideally with dedicated studies and prospective randomized trials.

Finally, in viable patients whose cardiac function recovery is highly unlikely, MCS should be considered as a bridge to OHT. The limited data of VSR patients undergoing OHT favors improved outcomes when compared to conventional surgical or percutaneous repair. However, OHT is limited by local access, time to transplant, and resource allocation.

CONCLUSIONS

VSR continues to be a challenging complication of AMI, with a poor prognosis that remains unaffected by advances in management. Despite efforts to improve outcomes, through numerous surgical repair technique iterations and novel percutaneous closure strategies, the odds have long favored mortality over survival, particularly among those with CS. The increasing use of MCS devices, institution of shock teams, and access to advance heart failure centers has created an air of optimism as we look to the future of post-AMI VSR management. However, collective efforts are necessary to integrate these emerging therapies into new treatment algorithms of post-MI VSR. Only through a collaborative multidisciplinary effort that prioritizes early, aggressive protocolized interventions and uses early involvement of tertiary referral centers will we improve outcomes for this condition.

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REFERENCES

1. Elbadawi A, Elgendy IY, Mahmoud K, et al. Temporal trends and outcomes of mechanical complications in patients with acute myocardial infarction. *J Am Coll Cardiol Intv*. 2019;12:1825–1836.

2. Figueras J, Alcalde O, Barrabes JA, et al. Changes in hospital mortality rates in 425 patients with acute ST-elevation myocardial infarction and cardiac rupture over a 30-year period. *Circulation*. 2008;118:2783–2789.

3. Matteucci M, Ronco D, Corazzari C, et al. Surgical repair of postinfarction ventricular septal rupture: systematic review and meta-analysis. *Ann Thorac Surg*. 2021;112:326–337.

4. Moreyra AE, Huang MS, Wilson AC, et al. Trends in incidence and mortality rates of ventricular septal rupture during acute myocardial infarction. *Am J Cardiol*. 2010;106:1095–1100.

5. French JK, Hellkamp AS, Armstrong PW, et al. Mechanical complications after percutaneous coronary intervention in ST-elevation myocardial infarction (from APEX-AMI). *Am J Cardiol*. 2010;105:59–63.

6. Crenshaw BS, Granger CB, Birnbaum Y, et al. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. GUSTO-I (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) Trial Investigators. *Circulation*. 2000;101:27–32.

7. Yip HK, Fang CY, Tsai KT, et al. The potential impact of primary percutaneous coronary intervention on ventricular septal rupture complicating acute myocardial infarction. *Chest*. 2004;125:1622–1628.

8. Ronco D, Matteucci M, Kowalewski M, et al. Surgical treatment of postinfarction ventricular septal rupture. *JAMA Netw Open*. 2021;4:e2128309.

9. Arnaoutakis GJ, Zhao Y, George TJ, Sciortino CM, McCarthy PM, Conte JV. Surgical repair of ventricular septal defect after myocardial infarction: outcomes from the Society of Thoracic Surgeons National Database. *Ann Thorac Surg*. 2012;94:436–443. discussion 443-44.

10. Giblett JP, Matetic A, Jenkins D, et al. Post-infarction ventricular septal defect: percutaneous or surgical management in the UK national registry. *Eur Heart J*. 2022;43:5020–5032.

11. Sanchez Vega JD, Alonso Salinas GL, Vieitez Florez JM, et al. Temporal trends in postinfarction ventricular septal rupture: the CIVIAM Registry. *Rev Esp Cardiol (Engl Ed)*. 2021;74:757–764.

12. Yang X, Yu Z, Wang Y, Ding Y, Ni R, Xiao P. Transcatheter closure for postinfarction ventricular septal defect: a meta-analysis of the current evidence. *J Card Surg*. 2021;36:4625–4633.

13. Damluji AA, van Diepen S, Katz JN, et al. Mechanical complications of acute myocardial infarction: a scientific statement from the American Heart Association. *Circulation*. 2021;144:e16–e35.

14. Ronco D, Matteucci M, Ravaux JM, et al. Mechanical circulatory support as a bridge to definitive treatment in post-infarction ventricular septal rupture. *J Am Coll Cardiol Intv*. 2021;14:1053–1066.

15. Perez-Villa B, Cubeddu RJ, Brozzi N, Sleiman JR, Navia J, Hernandez-Montfort J. Transition to heart transplantation in post-myocardial infarction ventricular septal rupture: a systematic review. *Heart Fail Rev*. 2023;28:217–227.

16. Patane F, Zingarelli E, Sansone F, Rinaldi M. Acute ventricular septal defect treated with an Impella recovery as a 'bridge therapy' to heart transplantation. *Interact Cardiovasc Thorac Surg*. 2007;6:818–819.

17. Pascual I, Lopez F, Hernandez-Vaquero D, Avanzas P, Silva J, Moris C. Circulatory support with extracorporeal membrane oxygenation system as a bridge to heart transplantation in complex postinfarction ventricular septal rupture. *Rev Esp Cardiol (Engl Ed)*. 2016;69:617–619.

18. Honda S, Asaumi Y, Yamane T, et al. Trends in the clinical and pathological characteristics of cardiac rupture in patients with acute myocardial infarction over 35 years. *J Am Heart Assoc*. 2014;3:e000984.

19. Lopez-Sendon J, Gurfinkel EP, Lopez de Sa E, et al. Factors related to heart rupture in acute coronary syndromes in the Global Registry of Acute Coronary Events. *Eur Heart J*. 2010;31:1449–1456.

20. Menon V, Webb JG, Hillis LD, et al. Outcome and profile of ventricular septal rupture with cardiogenic shock after myocardial infarction: a report from the SHOCK Trial Registry. Should we emergently revascularize occluded coronaries in cardiogenic shock? *J Am Coll Cardiol*. 2000;36:1110–1116.

21. De Rosa S, Spaccarotella C, Basso C, et al. Reduction of hospitalizations for myocardial infarction in Italy in the COVID-19 era. *Eur Heart J*. 2020;41:2083–2088.

22. Lai PH, Lancet EA, Weiden MD, et al. Characteristics associated with out-of-hospital cardiac arrests and resuscitations during the novel coronavirus disease 2019 pandemic in New York City. *JAMA Cardiol*. 2020;5:1154–1163.

23. Singh S, Fong HK, Mercedes BR, Serwat A, Malik FA, Desai R. COVID-19 and out-of-hospital cardiac arrest: a systematic review and meta-analysis. *Resuscitation*. 2020;156:164–166.

24. De Filippo O, D'Ascenzo F, Angelini F, et al. Reduced rate of hospital admissions for ACS during Covid-19 outbreak in northern Italy. *N Engl J Med*. 2020;383:88–89.

25. Rashid Hons M, Gale Hons CP, Curzen Hons N, et al. Impact of coronavirus disease 2019 pandemic on the incidence and management of out-of-hospital cardiac arrest in patients presenting with acute myocardial infarction in England. *J Am Heart Assoc*. 2020;9:e018379.

26. Bhatt AS, Moscone A, McElrath EE, et al. Fewer hospitalizations for acute cardiovascular conditions during the COVID-19 pandemic. *J Am Coll Cardiol*. 2020;76:280–288.

27. Mesnier J, Cottin Y, Coste P, et al. Hospital admissions for acute myocardial infarction before and after lockdown according to regional prevalence of COVID-19 and patient profile in France: a registry study. *Lancet Public Health*. 2020;5:e536–e542.

28. Metzler B, Siostrzonek P, Binder RK, Bauer A, Reinstadler SJ. Decline of acute coronary syndrome admissions in Austria since the outbreak of COVID-19: the pandemic response causes cardiac collateral damage. *Eur Heart J*. 2020;41:1852–1853.

29. Katsoularis I, Fonseca-Rodriguez O, Farrington P, Lindmark K, Fors Connolly AM. Risk of acute myocardial infarction and ischaemic stroke following COVID-19 in Sweden: a self-controlled case series and matched cohort study. *Lancet*. 2021;398:599–607.

30. Tsao CW, Aday AW, Almarzooq ZI, et al. Heart disease and stroke statistics-2022 update: a report from the American Heart Association. *Circulation*. 2022;145:e153–e639.

31. Primessnig U, Pieske BM, Sherif M. Increased mortality and worse cardiac outcome of acute myocardial infarction during the early COVID-19 pandemic. *ESC Heart Fail*. 2021;8:333–343.

32. Kubica J, Ostrowska M, Stolarek W, et al. Impact of COVID-19 pandemic on acute heart failure admissions and mortality: a multicentre study (COV-HF-SIRIO 6 study). *ESC Heart Fail*. 2022;9:721–728.

33. D'Ascenzo F, De Filippo O, Borin A, et al. Impact of COVID-19 pandemic and infection on in hospital survival for patients presenting with acute coronary syndromes: a multicenter registry. *Int J Cardiol*. 2021;332:227–234.

34. Riley RF, Kereiakes DJ, Mahmud E, Smith TD, Grines C, Henry TD. "Back to the Future" for STEMI?: the COVID-19 experience. *J Am Coll Cardiol Case Rep*. 2020;2:1651–1653.

35. Bakhshi H, Gattani R, Ekanem E, et al. Ventricular septal rupture and cardiogenic shock complicating STEMI during COVID-19 pandemic: an old foe re-emerges. *Heart Lung*. 2021;50:292–295.

36. Goraya MHN, Kalsoom S, Almas T, et al. Simultaneous left ventricular aneurysm and ventricular septal rupture complicating delayed STEMI presentation: a case-based review of post-MI mechanical complications amid the COVID-19 pandemic. *J Investig Med High Impact Case Rep*. 2021;9:23247096211031135.

37. Bryndza M, Legutko J, Kleczynski P, Kapelak B, Bartus K, Litwinowicz R. Impact of COVID-19 on the incidence of post-acute myocardial infarction mechanical complications. *Ann Cardiothorac Surg*. 2022;11:319–321.

38. Birnbaum Y, Fishbein MC, Blanche C, Siegel RJ. Ventricular septal rupture after acute myocardial infarction. *N Engl J Med*. 2002;347:1426–1432.

39. Vargas-Barrón J, Molina-Carrion M, Romero-Cardenas A, et al. Risk factors, echocardiographic patterns, and outcomes in patients with acute

ventricular septal rupture during myocardial infarction. *Am J Cardiol*. 2005;95:1153–1158.

40. Kim SD. Anatomy of the septal perforating arteries of the heart. *Anat Cell Biol*. 2019;52:236–241.

41. Dogra N, Puri GD, Thingnam SKS, et al. Early thrombolysis is associated with decreased operative mortality in postinfarction ventricular septal rupture. *Indian Heart J*. 2019;71:224–228.

42. Becker RC, Charlesworth A, Wilcox RG, et al. Cardiac rupture associated with thrombolytic therapy: impact of time to treatment in the Late Assessment of Thrombolytic Efficacy (LATE) study. *J Am Coll Cardiol*. 1995;25:1063–1068.

43. Jones BM, Kapadia SR, Smedira NG, et al. Ventricular septal rupture complicating acute myocardial infarction: a contemporary review. *Eur Heart J*. 2014;35:2060–2068.

44. Matteucci M, Ronco D, Massimi G, Di Mauro M, Lorusso R. Mechanical complications of acute myocardial infarction: from diagnosis to treatment. Article in Italian. *G Ital Cardiol (Rome)*. 2022;23:190–199.

45. Shahreyar M, Akinseye O, Nayyar M, Ashraf U, Ibebuogu UN. Post-myocardial infarction ventricular septal defect: a comprehensive review. *Cardiovasc Revasc Med*. 2020;21:1444–1449.

46. Cox FF, Morshuis WJ, Plokker HW, et al. Early mortality after surgical repair of postinfarction ventricular septal rupture: importance of rupture location. *Ann Thorac Surg*. 1996;61:1752–1757. discussion 1757-1758.

47. Edwards BS, Edwards WD, Edwards JE. Ventricular septal rupture complicating acute myocardial infarction: identification of simple and complex types in 53 autopsied hearts. *Am J Cardiol*. 1984;54:1201–1205.

48. Byrne RA, Rossello X, Coughlan JJ, et al. 2023 ESC Guidelines for the management of acute coronary syndromes. *Eur Heart J*. 2023;44:3720–3826.

49. Gueret P, Khalife K, Jobic Y, et al. Echocardiographic assessment of the incidence of mechanical complications during the early phase of myocardial infarction in the reperfusion era: a French multicentre prospective registry. *Arch Cardiovasc Dis*. 2008;101:41–47.

50. Dhaliwal S, Ducas R, Shuangbo L, et al. Multimodality cardiac imaging of a ventricular septal rupture post myocardial infarction: a case report. *BMC Res Notes*. 2012;5:583.

51. Gassenmaier T, Gorski A, Aleksic I, Deubner N, Weidemann F, Beer M. Impact of cardiac magnet resonance imaging on management of ventricular septal rupture after acute myocardial infarction. *World J Cardiol*. 2013;5:151–153.

52. Iyer S, Bauer T, Yeung M, et al. A heart team and multi-modality imaging approach to percutaneous closure of a post-myocardial infarction ventricular septal defect. *Cardiovasc Diagn Ther*. 2016;6:180–184.

53. Paul JF, Mace L, Caussin C, et al. Multirow detector computed tomography assessment of intraseptal dissection and ventricular pseudoaneurysm in postinfarction ventricular septal defect. *Circulation*. 2001;104:497–498.

54. Arzamendi D, Li CH, Serra A. Cardiac computed tomography-guided closure of ventricular septal defect secondary to myocardial infarction. *Rev Esp Cardiol (Engl Ed)*. 2015;68:626.

55. Ibanez B, James S, Agewall S, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: the Task Force for the Management of Acute Myocardial Infarction in Patients Presenting With ST-Segment Elevation of the European Society of Cardiology (ESC). *Eur Heart J*. 2018;39:119–177.

56. O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2013;61(4):485–510.

57. Lawton JS, Tamis-Holland JE, Bangalore S, et al. 2021 ACC/AHA/SCAI guideline for coronary artery revascularization: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2022;79(2):e21–e129.

58. Shibasaki I, Otani N, Saito S, et al. Overview of mechanical circulatory support for the management of post-myocardial infarction ventricular septal rupture. *J Cardiol*. 2023;81:491–497.

59. Liebelt JJ, Yang Y, DeRose JJ, Taub CC. Ventricular septal rupture complicating acute myocardial infarction in the modern era with mechanical circulatory support: a single center observational study. *Am J Cardiovasc Dis*. 2016;6:10–16.

60. Thiele H, Lauer B, Hambrecht R, et al. Short- and long-term hemodynamic effects of intraaortic balloon support in ventricular septal defect complicating acute myocardial infarction. *Am J Cardiol*. 2003;92:450–454.

61. Rob D, Spunda R, Lindner J, et al. A rationale for early extracorporeal membrane oxygenation in patients with postinfarction ventricular septal rupture complicated by cardiogenic shock. *Eur J Heart Fail*. 2017;19(suppl 2):97–103.

62. La Torre MW, Centofanti P, Attisani M, Patane F, Rinaldi M. Posterior ventricular septal defect in presence of cardiogenic shock: early implantation of the Impella Recover LP 5.0 as a bridge to surgery. *Tex Heart Inst J*. 2011;38:42–49.

63. Gregoric ID, Kar B, Mesar T, et al. Perioperative use of TandemHeart percutaneous ventricular assist device in surgical repair of postinfarction ventricular septal defect. *ASAIO J*. 2014;60:529–532.

64. Ancona MB, Regazzoli D, Mangieri A, Monaco F, De Bonis M, Latib A. Post-infarct ventricular septal rupture: early Impella implantation to delay surgery and reduce surgical risk. *Cardiovasc Interv Ther*. 2017;32:381–385.

65. Ariza-Sole A, Sanchez-Salado JC, Sbraga F, et al. The role of perioperative cardiorespiratory support in post infarction ventricular septal rupture-related cardiogenic shock. *Eur Heart J Acute Cardiovasc Care*. 2020;9:128–137.

66. McDonagh TA, Metra M, Adamo M, et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. *Eur Heart J*. 2021;42:3599–3726.

67. Jiritano F, Lo Coco V, Matteucci M, Fina D, Willers A, Lorusso R. Temporary mechanical circulatory support in acute heart failure. *Card Fail Rev*. 2020;6:e01.

68. O'Neill WW, Kleiman NS, Moses J, et al. A prospective, randomized clinical trial of hemodynamic support with Impella 2.5 versus intraaortic balloon pump in patients undergoing high-risk percutaneous coronary intervention: the PROTECT II study. *Circulation*. 2012;126:1717–1727.

69. Thiele H, Zeymer U, Neumann FJ, et al. Intraaortic balloon support for myocardial infarction with cardiogenic shock. *N Engl J Med*. 2012;367:1287–1296.

70. Pahuja M, Schrage B, Westermann D, Basir MB, Garan AR, Burkhoff D. Hemodynamic effects of mechanical circulatory support devices in ventricular septal defect. *Circ Heart Fail*. 2019;12:e005981.

71. Gambaro A, Rosenberg A, Galiatsou E, Stock UA. Pros and cons of different types of mechanical circulatory support device in case of postinfarction ventricular septal defect. *ASAIO J*. 2021;67:e110–e113.

72. Patane F, Centofanti P, Zingarelli E, Sansone F, La Torre M. Potential role of the Impella Recover left ventricular assist device in the management of postinfarct ventricular septal defect. *J Thorac Cardiovasc Surg*. 2009;137:1288–1289.

73. Kshettry VR, Salerno CT, Bank AJ. Risk of left ventricular assist device as a bridge to heart transplant following postinfarction ventricular septal rupture. *J Card Surg*. 1997;12:93–97.

74. Thiele H, Zeymer U, Akin I, et al. Extracorporeal life support in infarct-related cardiogenic shock. *N Engl J Med*. 2023;389:1286–1297.

75. Gregoric ID. Total artificial heart in patients with post-infarction ventricular septal defect. *Ann Cardiothorac Surg*. 2020;9:116–117.

76. Pitsis AA, Kelpis TG, Visouli AN, Bobotis G, Filippatos GS, Kremastinos DT. Left ventricular assist device as a bridge to surgery in post-infarction ventricular septal defect. *J Thorac Cardiovasc Surg*. 2008;135:951–952.

77. Faber C, McCarthy PM, Smedira NG, Young JB, Starling RC, Hoercher KJ. Implantable left ventricular assist device for patients with post-infarction ventricular septal defect. *J Thorac Cardiovasc Surg*. 2002;124:400–401.

78. Cooley DA, Belmonte BA, Zeis LB, Schnur S. Surgical repair of ruptured interventricular septum following acute myocardial infarction. *Surgery*. 1957;41:930–937.

79. Daggett WM, Guyton RA, Mundth ED, et al. Surgery for post-myocardial infarct ventricular septal defect. *Ann Surg*. 1977;186:260–271.

80. David TE, Dale L, Sun Z. Postinfarction ventricular septal rupture: repair by endocardial patch with infarct exclusion. *J Thorac Cardiovasc Surg*. 1995;110:1315–1322.

81. David TE, Armstrong S. Surgical repair of postinfarction ventricular septal defect by infarct exclusion. *Semin Thorac Cardiovasc Surg*. 1998;10:105–110.

82. Ronco D, Matteucci M, Massimi G, Di Mauro M, Lorusso R. Postinfarction ventricular septal rupture repair: is it just a matter of the surgical technique? *J Card Surg*. 2022;37:524–525.

83. Tabuchi N, Tanaka H, Arai H, et al. Double-patch technique for postinfarction ventricular septal perforation. *Ann Thorac Surg*. 2004;77:342–343.

84. Okamoto Y, Yamamoto K, Asami F, et al. Early and midterm outcomes of triple patch technique for postinfarction ventricular septal defects. *J Thorac Cardiovasc Surg*. 2016;151:1711–1716.

85. Lundblad R, Abdelnoor M. Surgery of post-infarction ventricular septal rupture: the effect of David infarct exclusion versus Daggett direct septal closure on early and late outcomes. *J Thorac Cardiovasc Surg*. 2014;148:2736–2742.

86. Vondran M, Wehbe MS, Etz C, et al. Mechanical circulatory support for early surgical repair of postinfarction ventricular septal defect with cardiogenic shock. *Artif Organs*. 2021;45:244–253.

87. Morimura H, Tabata M. Delayed surgery after mechanical circulatory support for ventricular septal rupture with cardiogenic shock. *Interact Cardiovasc Thorac Surg*. 2020;31:868–873.

88. Ronco D, Corazzari C, Matteucci M, et al. Effects of concomitant coronary artery bypass grafting on early and late mortality in the treatment of post-infarction mechanical complications: a systematic review and meta-analysis. *Ann Cardiothorac Surg*. 2022;11:210–225.

89. Ronco D, Matteucci M, Massimi G, Lorusso R. Concomitant surgical revascularization in post-infarction ventricular septal rupture and ventricular aneurysm repair: a straightforward indication or a prognostic factor? *J Card Surg*. 2022;37:2703–2705.

90. Horan DP, O'Malley TJ, Weber MP, et al. Repair of ischemic ventricular septal defect with and without coronary artery bypass grafting. *J Card Surg*. 2020;35:1062–1071.

91. Jeppsson A, Liden H, Johnsson P, Hartford M, Radegran K. Surgical repair of post infarction ventricular septal defects: a national experience. *Eur J Cardiothorac Surg*. 2005;27:216–221.

92. Pojar M, Harrer J, Omran N, Turek Z, Striteska J, Vojacek J. Surgical treatment of post-infarction ventricular septal defect: risk factors and outcome analysis. *Interact Cardiovasc Thorac Surg*. 2018;26:41–46.

93. Barker TA, Ramnarine IR, Woo EB, et al. Repair of post-infarct ventricular septal defect with or without coronary artery bypass grafting in the northwest of England: a 5-year multi-institutional experience. *Eur J Cardiothorac Surg*. 2003;24:940–946.

94. Mantovani V, Mariscalco G, Leva C, Blanzola C, Sala A. Surgical repair of post-infarction ventricular septal defect: 19 years of experience. *Int J Cardiol*. 2006;108:202–206.

95. Takahashi H, Arif R, Almashhoor A, Ruhparwar A, Karck M, Kallenbach K. Long-term results after surgical treatment of postinfarction ventricular septal rupture. *Eur J Cardiothorac Surg*. 2015;47:720–724.

96. Noguchi K, Yamaguchi A, Naito K, Yuri K, Adachi H. Short-term and long-term outcomes of postinfarction ventricular septal perforation. *Gen Thorac Cardiovasc Surg*. 2012;60:261–267.

97. Pang PY, Sin YK, Lim CH, et al. Outcome and survival analysis of surgical repair of post-infarction ventricular septal rupture. *J Cardiothorac Surg*. 2013;8:44.

98. Yam N, Au TW, Cheng LC. Post-infarction ventricular septal defect: surgical outcomes in the last decade. *Asian Cardiovasc Thorac Ann*. 2013;21:539–545.

99. Matteucci M, Kowalewski M, De Bonis M, et al. Surgical treatment of post-infarction left ventricular free-wall rupture: a multicenter study. *Ann Thorac Surg*. 2021;112:1186–1192.

100. Cox FF, Plokker HW, Morshuis WJ, Kelder JC, Vermeulen FE. Importance of coronary revascularization for late survival after postinfarction ventricular septal rupture. A reason to perform coronary angiography prior to surgery. *Eur Heart J*. 1996;17:1841–1845.

101. Skillington PD, Davies RH, Luff AJ, et al. Surgical treatment for infarct-related ventricular septal defects. Improved early results combined with analysis of late functional status. *J Thorac Cardiovasc Surg*. 1990;99:798–808.

102. Maltais S, Ibrahim R, Basmadjian AJ, et al. Postinfarction ventricular septal defects: towards a new treatment algorithm? *Ann Thorac Surg*. 2009;87:687–692.

103. Holzer R, Balzer D, Amin Z, et al. Transcatheter closure of postinfarction ventricular septal defects using the new Amplatzer muscular VSD Occluder: results of a U.S. registry. *Catheter Cardiovasc Interv*. 2004;61:196–201.

104. Calvert PA, Cockburn J, Wynne D, et al. Percutaneous closure of postinfarction ventricular septal defect: in-hospital outcomes and long-term follow-up of UK experience. *Circulation*. 2014;129:2395–2402.

105. Leung K, Bhansali HM, Ghiloni N, et al. Transseptal VSD closure in a patient with mechanical aortic and tricuspid valves. *J Am Coll Cardiol*. 2020;75(suppl 1):2372.

106. Meng W, Lin K, Zhang E. Perventricular closure on the beating heart: an effective hybrid approach in managing a postinfarction apical ventricular septal defect. *J Card Surg*. 2013;28:16–18.

107. Kamioka N, Lederman RJ, Greenbaum AB, et al. Postinfarction ventricular septal defect closure. *Circ Cardiovasc Interv*. 2019;12:e007788.

108. Kharge J, Parikh CJ, Suranagi MJ, Lakshmanasastry S, Srinivasa KH, Manjunath CN. Indicators and predictors of in-hospital mortality and survival in patients with ventricular septal rupture. *Am Heart J Plus*. 2022;13:100095.

109. Cummings RG, Califf R, Jones RN, Reimer KA, Kong YH, Lowe JE. Correlates of survival in patients with postinfarction ventricular septal defect. *Ann Thorac Surg*. 1989;47:824–830.

110. Giblett JP, Jenkins DP, Calvert PA. Transcatheter treatment of postinfarct ventricular septal defects. *Heart*. 2020;106:878–884.

111. Schlotter F, de Waha S, Eitel I, Desch S, Fuernau G, Thiele H. Interventional post-myocardial infarction ventricular septal defect closure: a systematic review of current evidence. *EuroIntervention*. 2016;12:94–102.

112. Flynn CD, Morris P, Manuel L, et al. Systematic review and meta-analysis of the mechanical complications of ischemic heart disease: papillary muscle rupture, left ventricle rupture and post-infarct ventricular septal defect. *Ann Cardiothorac Surg*. 2022;11:195–209.

113. Gill M, Bagshaw SM, McKenzie E, et al. Patient and family member-led research in the intensive care unit: a novel approach to patient-centered research. *PLoS One*. 2016;11:e0160947.

114. Naib T, Lahewala S, Arora S, Gidwani U. Palliative care in the cardiac intensive care unit. *Am J Cardiol*. 2015;115:687–690.

115. Goldfarb M, Bibas L, Burns K. Patient and family engagement in care in the cardiac intensive care unit. *Can J Cardiol*. 2020;36:1032–1040.

116. Ellis CJ, Parkinson GF, Jaffe WM, Campbell MJ, Kerr AR. Good long-term outcome following surgical repair of post-infarction ventricular septal defect. *Aust N Z J Med*. 1995;25:330–336.

117. Matteucci M, Ronco D, Kowalewski M, et al. Long-term results after surgical treatment of post-infarction mechanical complications: results from the CAUTION study. *Eur Heart J Qual Care Clin Outcomes*. Published online February 7, 2024. https://doi.org/10.1093/ehjqcco/qcae010

118. Sulzgruber P, El-Hamid F, Koller L, et al. Long-term outcome and risk prediction in patients suffering acute myocardial infarction complicated by post-infarction cardiac rupture. *Int J Cardiol*. 2017;227:399–403.

119. Yousef S, Sultan I, VonVille HM, Kahru K, Arnaoutakis GJ. Surgical management for mechanical complications of acute myocardial infarction: a systematic review of long-term outcomes. *Ann Cardiothorac Surg*. 2022;11:239–251.

120. Arnaoutakis GJ, Kilic A, Conte JV, et al. Longitudinal outcomes after surgical repair of postinfarction ventricular septal defect in the Medicare population. *Ann Thorac Surg*. 2020;109:1243–1250.

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